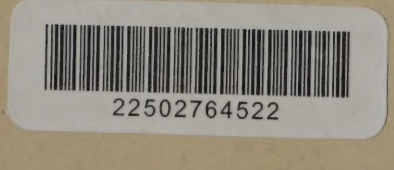

ARMY.

Second Report by the Director-General, Army Medical Service, on the Transmission of Enteric Fever by the "Chronic Carrier."

(*In continuation of Command Paper No.* 4,609.)

Presented to Parliament by Command of His Majesty.

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Second Report by the Director-General, Army Medical Service, on the Transmission of Enteric Fever by the "Chronic Carrier."

(*In continuation of Command Paper No.* 4,609.)

Introduction.

In the first report on "typhoid carriers" it was stated that recent studies on the saprophytic existence of the *B. typhosus* having shown that the life of this bacillus in soil and water does not exceed two months, Koch evolved the idea that in certain outbreaks of enteric fever man must be the chief transmitting agent of the infecting microbe. Investigations in Germany by Koch and his colleagues soon demonstrated that from 3 to 4 per cent. of typhoid convalescent patients excreted the *B. typhosus* for an indefinite period, *i.e.*, became "typhoid carriers." Impressed with Koch's results, the Indian Government established a depôt at Naini Tal for the convalescent patients of the 3rd, 5th, 7th and 8th divisions, and during 1908–1909 typhoid carrier cases were detected amongst these men and invalided to England. Thereupon the Army Medical Advisory Board took up the treatment of these carriers and, as stated in the first report, recommended that some cases should be treated by specific vaccines, each prepared from the strain of *B. typhosus* excreted by the particular patient, and other cases by lactic acid bacilli; methods now being tried by other authorities in civil life. Further details of the treatment of these carrier cases are now given.

FURTHER DETAILS OF THE TREATMENT OF THE "CARRIER CASES."

Treatment by Cultures of Lactic Acid Bacilli.

Gunner C.—Treatment was commenced on March 8th in the Queen Alexandra Military Hospital, and continued there until July 23rd. The dose of culture given at first was 25 c.c. daily; it was increased on March 12th to 50 c.c., on March 17th to 100 c.c., on April 6th to 200 c.c., on April 29th to 250 c.c., and on June 2nd to 300 c.c. daily.

Before treatment was commenced estimations were made of the numbers of *B. coli* and *B. typhosus;* the numbers of *B. coli* were found to vary from 207 to 4 millions per gramme of fæces and of *B. typhosus* from 190 to 14 millions per gramme. This enormous variation is probably explained by the varying consistency of the stools, some specimens containing more water than others. In order to obtain exactly comparable results, it would have been necessary to dry the stools, but this procedure would probably have injured the living bacteria. The estimations, however, gave a useful idea of the numbers being excreted at the time the treatment was begun. (*See* Chart I.)

Eight days after the lactic acid bacilli were given, there was a marked fall in the numbers of *B. coli* excreted, but there was not any similar fall in the numbers of *B. typhosus*. Since that time the numbers of *B. coli* have remained at a comparatively low level with the exception of occasional sharp rises. The numbers of *B. typhosus*, however, remained at a constantly high level, with occasional sharp falls, until the end of May. The lactic acid bacteria were known to be established in the stools during this period—but experiments *in vitro* showed that the strain of *B. typhosus* excreted by Gunner C. was much more resistant to the toxines of the lactic acid bacillus than other strains of the *B. typhosus*, notably that excreted by the next patient on the list, Private H.

During the month of June the excretion of *B. typhosus* appeared to be influenced by the treatment, as the numbers excreted fell to 9 millions and then to 200,000 per gramme of stool. From the 16th to the 25th of June the lactic acid bacilli could not

CHART I.

MILLIONS OF BACILLI PER GRAMME OF THE FAECES

MARCH 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31

APRIL 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30

MAY 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31

JUNE 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30

JULY 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24

AUGUST 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31

SEPTEMBER 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30

27·0 26·0 25·0 24·0 23·0 22·0 21·0 20·0 19·0 18·0 17·0 16·0 15·0 14·0 13·0 12·0 11·0 10·0 9·0 8·0 7·0 6·0 5·0 4·0 3·0 2·0 1·0 ·0

190 65 207 37

TREATMENT COMMENCED 25 C.C. DAILY

50 C.C. DAILY

100 C.C. DAILY

(ABSENT FROM HOSPITAL. NO TREATMENT DURING THREE DAYS.)

73 124 180 43 74 48 36 86 55 74 99

250 C.C. DAILY

300 C.C. DAILY

38 30 55 80 39 66 46 52 60

B.B. ISOLATED FROM FAECES

B.B. NOT FOUND IN FAECES

BLACK LINE —— B. COLI.
RED LINE B. TYPH.

15,000 | Less than 5,000 | 100,000 | 100,000 | 50,000 | 80,000 | 180,000

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Chart II.

MILLIONS OF BACILLI PER GRAMME OF THE FAECES

1 MARCH 1 APRIL 1 MAY 1 JUNE 1 JULY

TREATMENT COMMENCED. 25 C.C. DAILY.
50 C.C. DAILY.
100 C.C. DAILY.
200 C.C. DAILY.
250 C.C. DAILY.
TREATMENT STOPPED

B.B. ISOLATED FROM FAECES
B.B. NOT FOUND IN FAECES

43
1000
300

BLACK LINE ——— B. COLI.
RED LINE ——— B. TYPH.

NIL
Less than 5000
Less than 200
4,600
30,000
Less than 200
NIL

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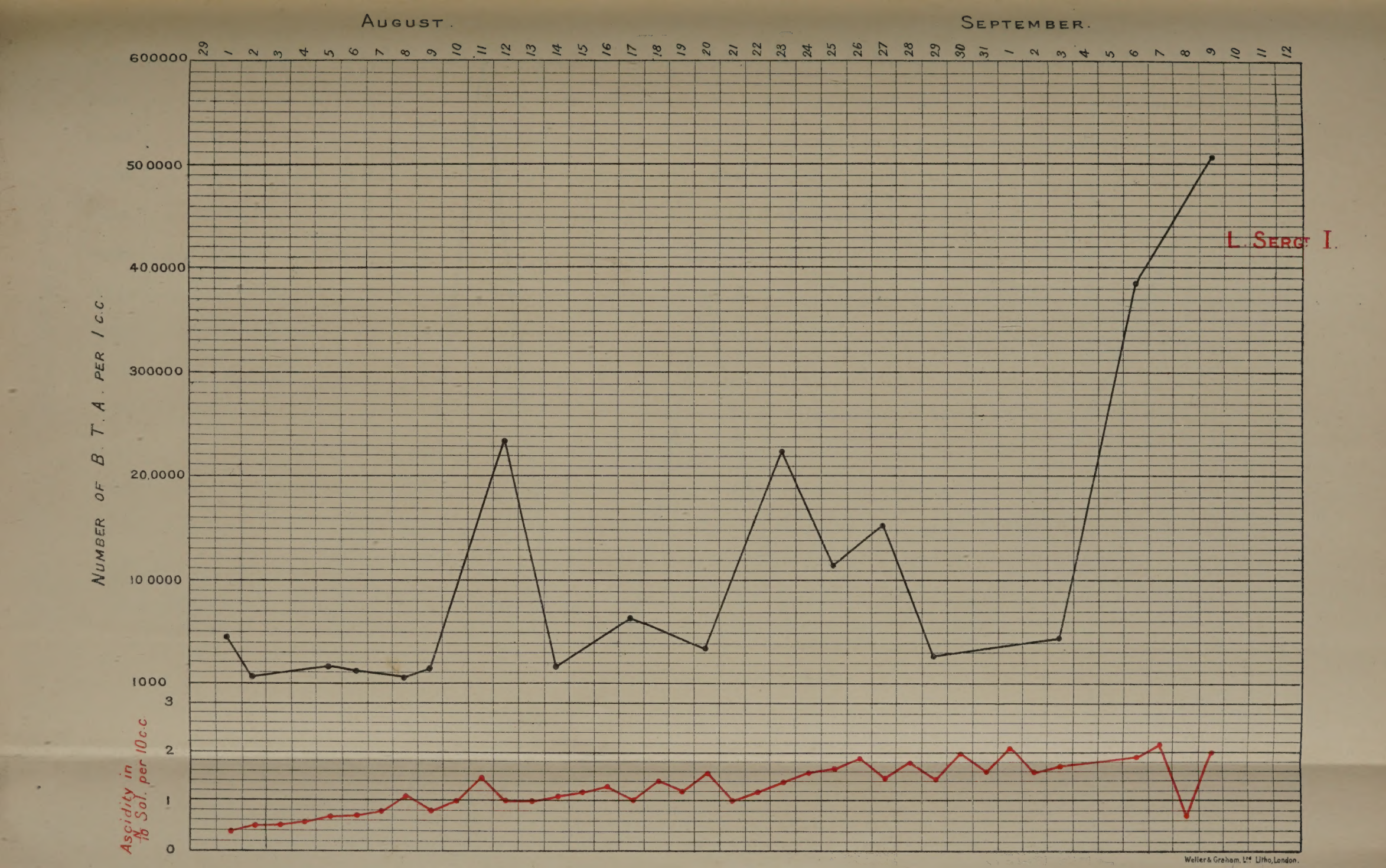

August.
September.
29 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 1 2 3 4 5 6 7 8 9 10 11 12
600000
50 0000
40 0000
300000
20,0000
10 0000
1000
Number of B. T. A. per 1 c.c.
3
2
1
0
Ascidity in N/10 Sol. per 10 c.c.
L. Sergt I.
Weller & Graham, Ltd Litho, London.

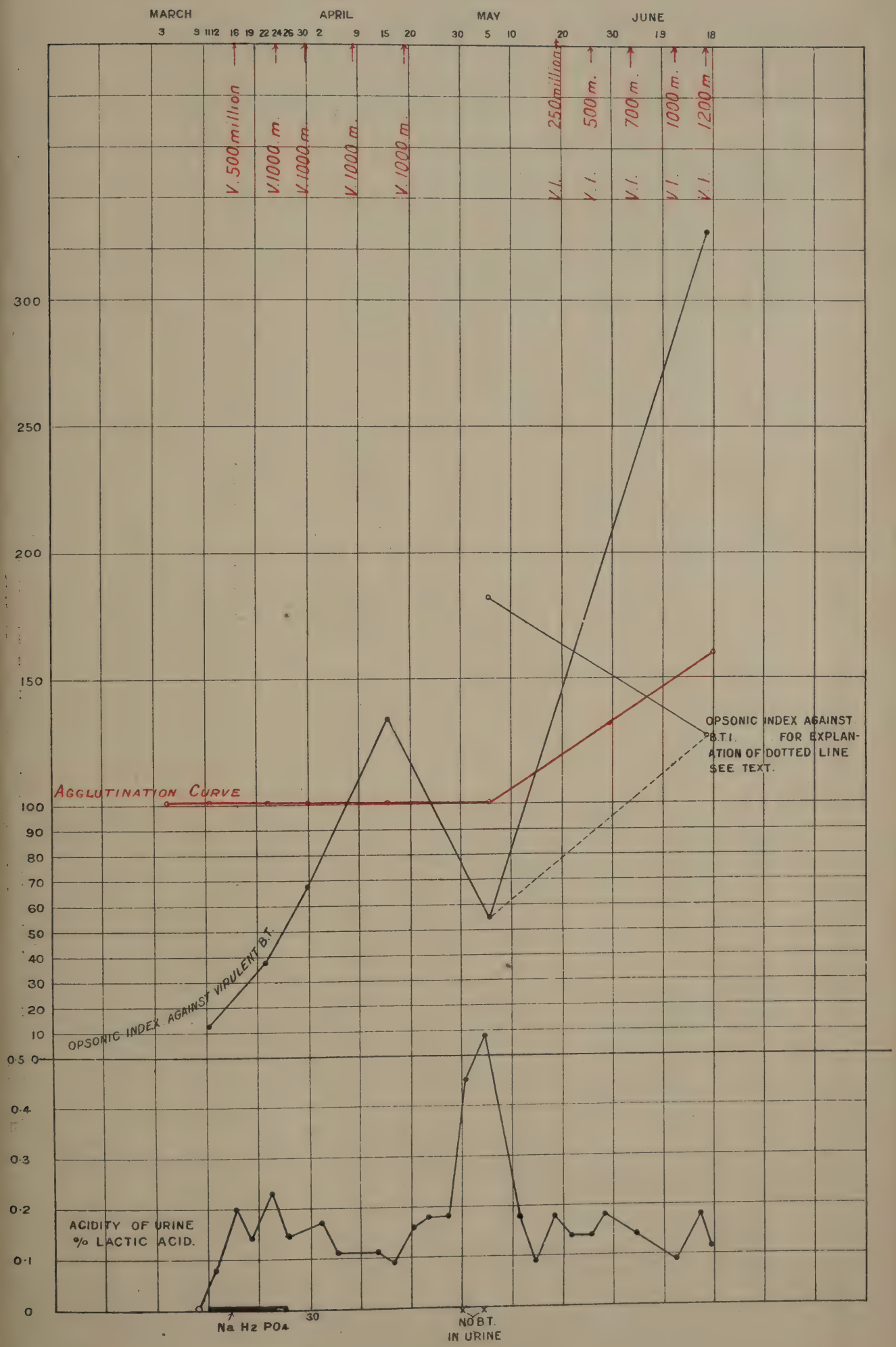

AUGUST.
SEPTEMBER.
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 1 2 3 4 5 6 7 8 9 10 11 12 13 14
15000
14000
13000
12000
11000
10000
9000
8000
7000
6000
5000
4000
3000
2000
1000
0
NUMBER OF TYPHOID BACILLI PER 1 C.C OF URINE.
B R S.
6.8
4
3
2
1
0
Acidity in
No Alk. per 10c.c Urine
Weller & Graham. Ltd Litho, London.

L'Sgt. I.

Chart V.

Doses of Vaccine. Curves of Agglutinins and of Opsonins.

Acidity of Urine.



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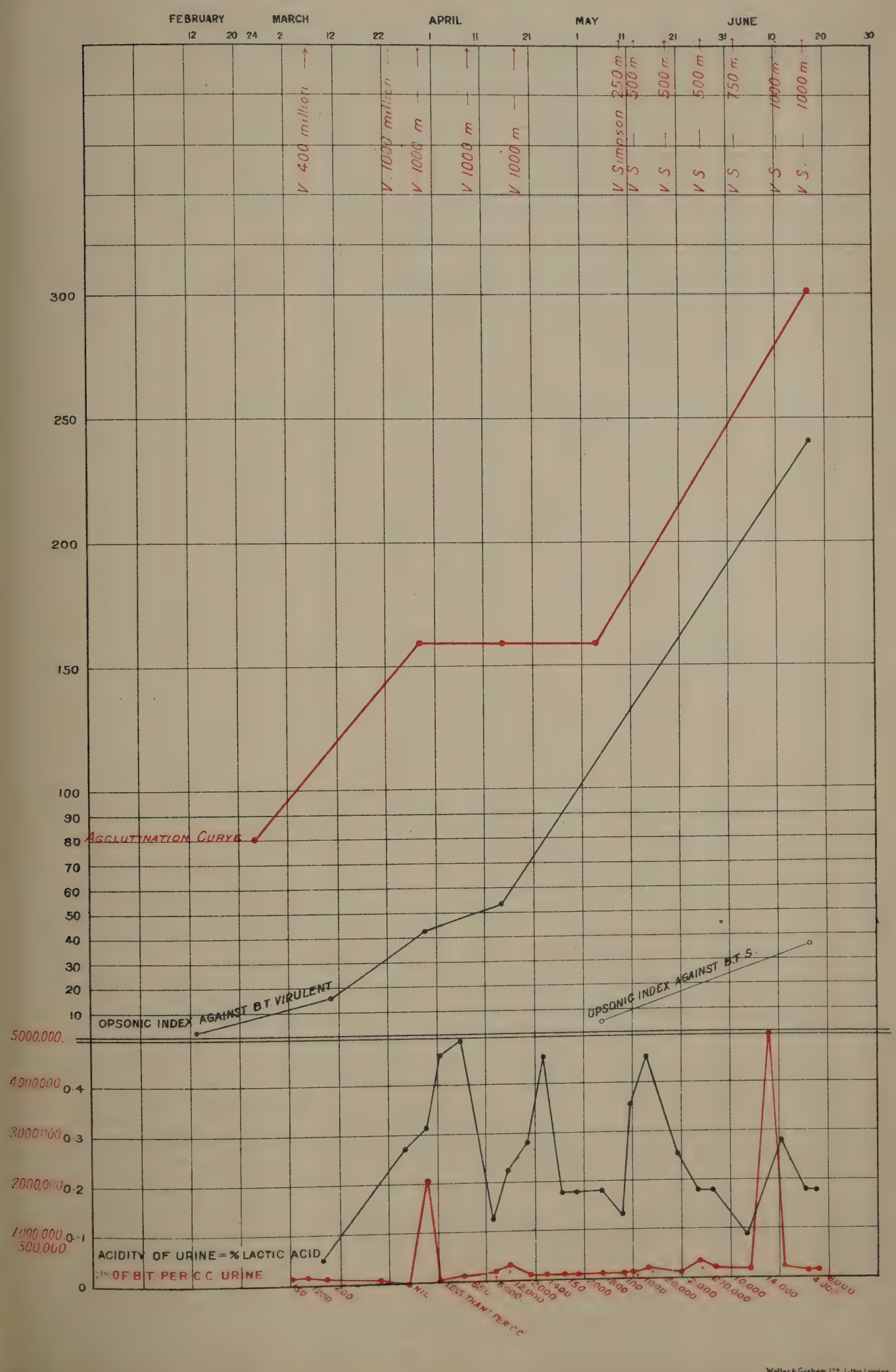
S.

CHART VI.

DATES OF VACCINATION. CURVES OF AGGLUTININS AND OF OPSONINS

ACIDITY OF URINE. EXCRETION OF B. TYPHOSUS IN URINE.



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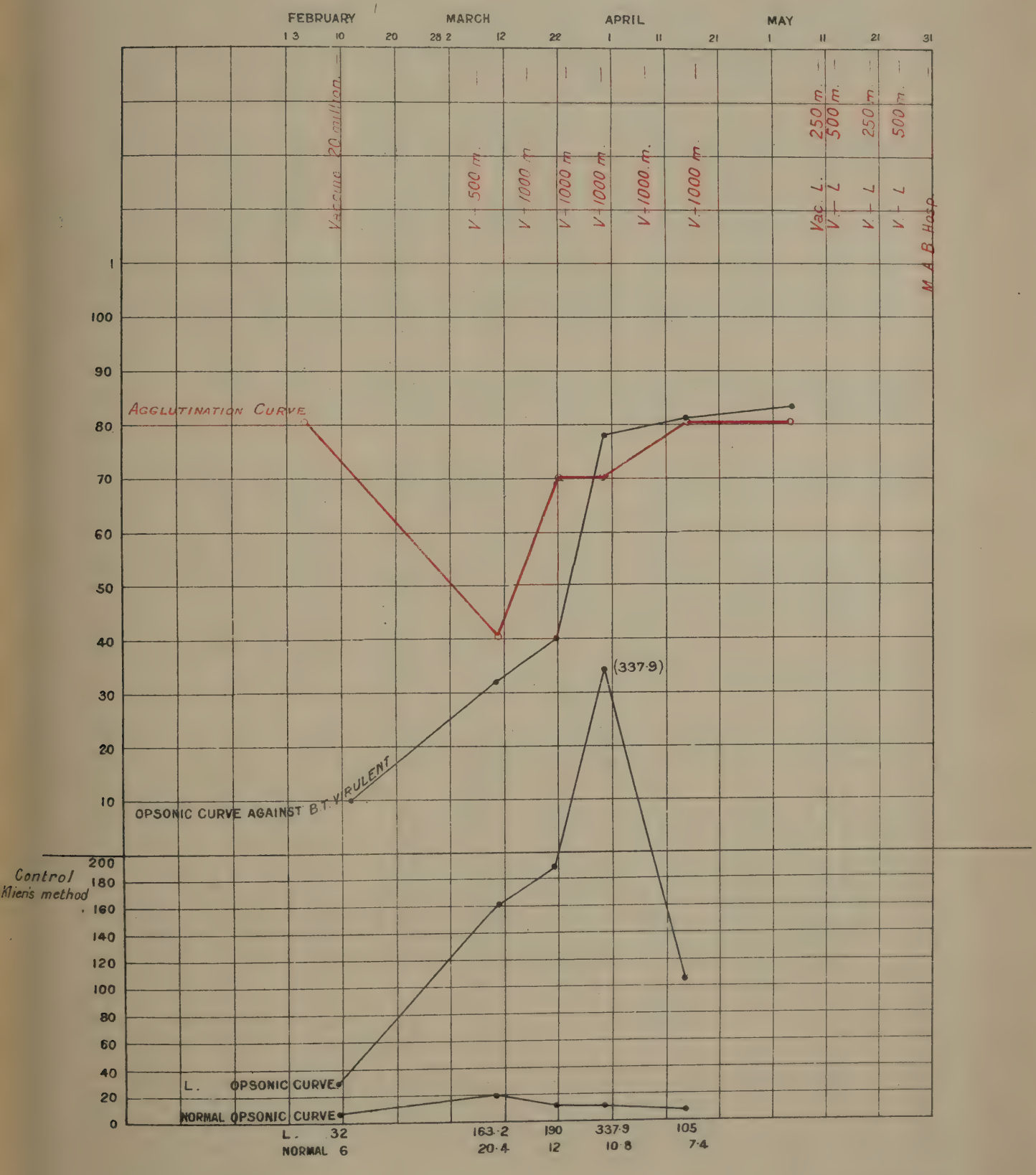
PTE. L——.

CHART VII.

DATES OF VACCINATION, CURVES OF OPSONINS AND OF AGGLUTININS



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be found in the excreta, and on the 29th June the numbers of typhoid bacilli excreted were found to be at least 30 millions per gramme of stool. On enquiring into the cause for this condition of things it was found that several times during the month of June the man had not taken the prescribed doses of lactic acid bacilli. In July the lactic acid bacilli were again found in the stools and the excretion of typhoid bacilli fell to 200,000. In the middle of July a sharp rise in the excretion occurred, which was followed by a fall to one million at the end of the month.

On July 23rd the man was transferred to Netley, where the same treatment was continued. On August 9th, for the first time since the man came under treatment, no typhoid bacilli could be found in the excreta; the dosage with lactic acid bacilli was then omitted. It was hoped that the man was on the high road to a cure, but unfortunately in the middle of August some 60 million typhoid bacilli again appeared in the stools. Lactic acid treatment was again commenced and the excretion of typhoid bacilli has once more fallen to about 10 millions.

It will be remembered that before the lactic acid treatment was commenced the excretion of typhoid bacilli varied from 190 to 14 millions. The high level of excretion was maintained during May and June, when presumably the lactic acid bacilli were producing toxines which experiments *in vitro* had shown would act slowly on the strain of typhoid bacillus present. The rapid variations in the excretion of typhoid bacilli following on the omission of the lactic acid bacilli during the later phases of the treatment, and the actual extinction of the typhoid bacilli on August 9th seem to justify the belief that the lactic acid treatment has a distinct influence on the excretion of typhoid bacilli and hold out a distinct prospect of cure when the treatment is continued without intermission.

Private H.—The results of the quantitative examination of the fæces of this man are given in Chart II. Before treatment was begun he was excreting the *B. typhosus* regularly, and the numbers varied between 4·5 and 3 million bacilli per gramme of stool.

Treatment was commenced on March 8th. At first 25 c.c. of lactic acid bacilli culture were given; the dose was increased on March 12th to 50 c.c., on March 17th to 100 c.c., on April 3rd to 200 c.c., and on April 29th to 250 c.c. daily.

No change of any importance in the excretion of typhoid bacilli occurred until March 26th, when they could not be found in the stools. The lactic acid bacilli, however, were then found, for the first time, in the excreta. On March 30th the typhoid bacilli reappeared in the stools and continued in practically undiminished numbers until April 30th, fifty-three days after the commencement of the lactic acid treatment, when they again disappeared.

Dosage with lactic acid bacilli was omitted on May 28th, and since April 30th typhoid bacilli have not appeared again in the stools. The excreta were regularly examined until July, when the man was discharged to duty. It was noted that, 14 days after the omission of the lactic acid treatment, there was a rapid rise in the excretion of *B. coli*, showing that the inhibiting effects of the lactic acid bacilli were disappearing, but no signs of the *B. typhosus* could be found.

It should be noted that experiments *in vitro* showed that the strain of *B. typhosus* excreted by Private H. was much more susceptible to the influence of the toxines of the lactic acid bacilli than the strain excreted by Gunner C., the first case submitted to the lactic acid treatment. The feeble resistance of the strain of *B. typhosus* would seem to explain the more rapid and favourable result obtained in Private H.'s case.

Though discharged to duty, arrangements will be made for the examination of Private H. at stated intervals, so as to determine the permanency of the cure.

Treatment by Anti-Typhoid Vaccine.

Preliminary observations.—It was desired to obtain some idea of the immune substances present in the blood of each of the seven cases, and estimations were therefore made of the agglutinins and of the opsonins.

Agglutinins.—The following were the reactions obtained with a virulent laboratory strain of *B. typhosus*:—

Private S.—Incomplete in a dilution of 1–160.

Lance-Serjeant I.—Complete in a dilution of 1–100, not in 1–120.

Private L.—Complete in a dilution of 1–80.

Bombardier S.—Incomplete in a dilution of 1–80.
Private O'N.—Complete in a dilution of 1–40.
Gunner C.—Incomplete in a dilution of 1–20.
Private H.—Incomplete in a dilution of 1–20.

A further series of observations was then made to see whether the sera of these men behaved differently when tested with their individual strains of *B. typhosus*; at the same time they were tested against a non-virulent laboratory strain. The results are tabulated below :—

	B. typhosus. Non-virulent.					*B. typhosus.* Virulent.					*B. typhosus.* Specific Strain.				
Serum dilutions	10	20	40	80	160	10	20	40	80	160	10	20	40	80	160
Gunner C...	±	+	±	−	−	+	±	±	−	−	±	−	−	−	−
Private L...	..	+	+	±	−	+	+	+	+	−	+	+	+	±	−
Bombardier S.	+	+	+	±	..	+	+	+	±	..	+	+	+	±	..

+ Signifies complete agglutination. ± Signifies incomplete agglutination.
− Signifies no agglutination.

The serum of Lance-Serjeant I. was tested against the virulent laboratory strain and his own specific strain, with the following result :—

Dilution.	*Virulent.*	*Specific.*
10	+	Not tested.
20	+	,,
40	+	,,
80	+	,,
100	+	±
200	−	−

The serum of Private H. tested against the two laboratory strains gave a complete reaction in 1–40 and incomplete in 1–80 with the non-virulent laboratory strain; with the virulent the reaction was incomplete in the 1–20 dilution.

Opsonins.—The first observations were made with the sera of Private L. and Gunner C. Two methods were employed :—

(i) Klien's method, in which the dilution of serum which will give a phagocytosis per cell of 0·5 is estimated.

(ii) The heated serum of the patient was diluted to 1–4 and added to equal volumes of washed cells and bacterial emulsion. The phagocytic index of the immune sera was then obtained by dividing the average phagocytosis per cell obtained with the patient's serum by that obtained with the normal serum.

Each serum was tested with the above-mentioned virulent strain and the non-virulent laboratory strain, and also with the specific strain derived from the patient himself.

The control normal serum in each experiment was tested with the two laboratory strains only.

The following tables give the results :—

1. *Klien's method.*—The numbers indicate the dilution of serum which was necessary to give a phagocytosis of 0·5 bacterium per cell.

Serum.	*B. typhosus.* Non-virulent.	*B. typhosus.* Virulent.	*B. typhosus.* Specific.
Normal	About 28	6	—
Gunner C... ..	,, 96	20	9
Private L... ..	96	96	70

2. *Diluted heated serum.*—The numbers indicate the average number of bacilli phagocyted by each cell.

Serum.	*B. typhosus.* Non-virulent.	*B. typhosus.* Virulent.	*B. typhosus.* Specific.
Normal	0·3	0·2	—
Gunner C. . ..	0·6*	0·5	0·45
Private L.. ..	0·7*	2·0	1·25

* These slides were very difficult to count, there being many "ghost forms" and evidence of lysis.

An examination of these tables will show that they agree in the main, with the exception of the figures marked *.

The following points were deduced from these experiments :—

(1) That the virulent strain of *B. typhosus* would be more reliable for future work than the non-virulent strain, which was too easily phagocyted and bacteriolysed.

(2) That the specific strains were less easily phagocyted than the laboratory strains. This corresponds with the observations on the agglutinins, especially with regard to Gunner C.

(3) That the ratio of the phagocytic power of the three sera, when tested against the virulent laboratory strain, was very similar with both methods. *Klien.*—Normal, Gunner C., Private L. : 1, 3, 16. *Heated serum.*—Normal, Gunner C., Private L. : 1, 2·5, 10. Compare this with the agglutination reaction: Gunner C. = 1–20 ; Private L. = 1–80.

On account of the labour involved in Klien's method, all the subsequent opsonic observations were made with the heated diluted serum and, where possible, a control observation was made by Klien's method with one of the sera (L.'s), the strain used being the virulent laboratory one.

The sera of Lance-Serjeant I. and Bombardier S. were also tested for opsonins against the laboratory strain, and gave indices of 13 and 2·25 respectively, compared with Private L. and Gunner C., 10 and 2·5 respectively.

Treatment by vaccine.—The vaccine used was the prophylactic vaccine prepared at the Royal Army Medical College from the strain mentioned above as non-virulent. It was administered in doses rising to 1,000 million, at intervals of a week to ten days, until the middle of April. At this time as the cases did not show any sign of improvement it was considered advisable, in view of the indication afforded by agglutination and opsonic experiments, to substitute a vaccine prepared from the carrier's own strain of *B. typhosus.* Vaccines were accordingly prepared from the strains of Private L., Lance-Serjeant I. and Bombardier S., and administered during the months of May and June.

The particulars of the cases are as follows (*vide* Charts) :—

Private L.—Vaccine (prophylactic)—

Administered 10th February.	20 million.	Local reaction.
8th March.	500 ,,	,, ,, and malaise.
†16th ,,	1,000 ,,	Slight local reaction and malaise.
24th ,,	1,000 ,,	
31st ,,	1,000 ,,	
9th April.	1,000 ,,	
19th ,,	1,000 ,,	

Vaccine (specific)—

Administered 10th May.	250 ,,	No reaction.
12th ,,	500 ,,	Severe local reaction.
20th ,,	250 ,,	
26th ,,	500 ,,	Very slight reaction.

On May 31st he contracted diphtheria and was transferred to the Metropolitan Asylums Board Hospital.

† Region of gall bladder.

Observations on the agglutinins and opsonins were made from February 10th to May 5th. No observations could be made after the administration of the specific vaccine on account of his removal.

Chart VII indicates the opsonic curve and shows that the agglutination remained stationary.

The double observation of the opsonic content serves as a control, but unfortunately the control shows a marked discrepancy on April 15th.

The excretion of germs in the stools showed an intermission dating from April 16th for a period of several weeks.

Bombardier S.—Vaccine (prophylactic)—

Administered	8th March.	400	million.	Local reaction, malaise.
	24th ,,	1,000	,,	Very slight reaction.
	30th ,,	1,000	,,	,, ,, ,,
	9th April.	1,000	,,	
	19th ,,	1,000	,,	
Vaccine (specific)—				
Administered	10th May.	250	,,	No reaction.
	12th ,,	500	,,	Severe local reaction 2 days.
	20th ,,	500	,,	Slight reaction.
	27th ,,	500	,,	,, ,,
	3rd June.	750	,,	,, ,,
	11th ,,	1,000	,,	,, ,,
	17th ,,	1,000	,,	,, ,,

Effect on immune substances. (*See* Chart VI.)

Agglutinins rose from 1–80 to 1–160 and then remained stationary during administration of the prophylactic vaccine, but after the use of the specific vaccine they rose to 1–300, when tested against the virulent laboratory strain.

Opsonins showed a steady rise all through, as tested against the virulent laboratory strain. There was also a marked increase latterly when tested against his own strain of *B. typhosus.*

The Chart also gives the acidity of the urine and the number of typhoid bacilli excreted in each c.c. of urine.

Lance-Serjeant I.—Vaccine (prophylactic)—

Administered	16th March.	500	million.	Very slight reaction.
	24th ,,	1,000	,,	,, ,, ,,
	30th ,,	1,000	,,	,, ,, ,,
	9th April.	1,000	,,	,, ,, ,,
	19th ,,	1,000	,,	,, ,, ,,
Vaccine (specific)—				
Administered	20th May.	250	,,	Moderate reaction.
	26th ,,	500	,,	,, ,,
	3rd ,,	700	,,	,, ,,
	11th ,,	1,000	,,	,, ,,
	17th ,,	1,200	,,	,, ,,

For observations on agglutinins and opsonins, *see* Chart V.

The *agglutinins* remained stationary all through the period of administration of the prophylactic vaccine (viz., 1–100), but after the specific vaccine was given they rose to 1–160, when tested against the virulent laboratory strain.

The *opsonins* show a steady rise until the observation of May 5th, on which date the phagocytic ratio was only 55 as compared with 134 on April 15th.

The curve at the foot of the Chart is interesting and shows the acidity of the urine as per cent. of lactic acid. The black line marked NaH_2PO_4 represents the time during which acid sodium phosphate was given and the increase in the acidity is well marked.

There was, however, no cessation of excretion of bacilli in the urine. On April 30th and May 4th the acidity rose to the normal, and on these days the urine was free from bacilli; it was also quite clear. At all other times the urine was cloudy.

With the reappearance of the bacilli on May 11th the acidity fell to its former level and the urine became cloudy.

It will be remarked that the phagocytic ratios obtained in all these experiments are unusually high. This is to be explained by the fact that the serum was diluted four times and consequently the phagocytosis by the normal serum was so slight at times as

to be almost not significant. The other alternative (conditions favouring phagocytosis with the normal serum) meant that the immune sera produced such an enormous phagocytosis as to render an accurate count a practical impossibility. The normal serum used was obtained from the same individual throughout.

Treatment by Administering Intestinal Antiseptics.

Gunner S. was treated in this manner, and it was hoped that by diminishing the gastric and intestinal food-content, excessive dilution of the drugs employed would be avoided. The patient consented to try a low diet for a time, and only milk and eggs were given. Urotropin was then administered in gradually increasing doses, beginning with 30 and ending with 48 grains *per diem*. Two teaspoonfuls of castor oil were also given daily. As will be seen by the Chart the numbers of typhoid bacilli fell, being only 2,000,000 per gramme of stool at the end of seven days. The patient's weight, however, diminished rapidly, and it was deemed advisable to give him a full diet. The number of bacteria at once rose. Jey's intestinal palatinoids were then tried, but produced no effect on the excretion of bacilli.

The man is now being treated with cultures of lactic acid bacilli.

Treatment by Raising the Acidity in the Urine.

Experiments *in vitro* having shown that the *B. typhosus* is unable to live in a medium containing acid to the extent of + 50 (*i.e.*, 50 c.c. of normal alkali required to neutralize one litre), it was determined to treat Bombardier S. and Lance-Serjeant I. with sodium benzoate and acid phosphate of soda, in the hope of raising the acidity of the urine in each case to the lethal point for the *B. typhosus*. Thirty grains of each of the drugs were given to each of the men three times a day.

Under this treatment the acidity of the urine of Bombardier S. rose during the first week from +20 to +33, and the excretion of typhoid bacilli fell from 2,700 per c.c. to 90 per c.c. On August 14th only seven bacilli per c.c. were isolated from the urine, and a successful termination of the case was anticipated. Unfortunately, in spite of the treatment, the acidity of the urine began to fall, and a corresponding increase in the excretion of typhoid bacilli at once took place.

The dose of each of the drugs was then increased and the acidity of the urine rose to + 35, but this was not maintained, and at the present time the acidity equals + 11 and the numbers of typhoid bacilli in the urine are practically the same as when the treatment was commenced.

The urine of Lance-Serjeant I. contained many more typhoid bacilli than that of Bombardier S., 55,800 bacilli per c.c. being counted when the treatment was begun. During the first week of treatment with sodium benzoate and acid phosphate of soda, the acidity of the urine was very low and typhoid bacilli were excreted in large numbers. In the following week the acidity arose to + 11, but the excretion of bacilli was uninfluenced ; indeed, on one day, August 11th, as many as 235,800 bacilli per c.c. were counted. Under the influence of large doses of the drugs, the acidity of the urine gradually rose to + 20 in the fourth week of treatment, yet more than 20,000 typhoid bacilli per c.c. were still excreted. At the present time the acidity of the urine equals + 17, and 44,320 typhoid bacilli are being excreted. (*See* Charts III and IV.)

Treatment by Exposure to X-Rays.

Private L., who suffered from occasional attacks of inflammation of the gall-bladder, and failed to respond to treatment with anti-typhoid vaccine, has been treated by exposure to X-rays. He has been X-rayed three times weekly, the gall-bladder alone being exposed for three minutes to an 8-inch tube. Since the commencement of this treatment typhoid bacilli have not been found in the fæces. Without more prolonged observation of this man it is impossible to say whether he is cured, as in his case the excretion of typhoid bacilli has been markedly intermittent, and on a former occasion an intermission of four weeks occurred.

Probable Reasons why the Treatment before mentioned has not Proved Effective.

In order to kill the typhoid bacillus it is essential that the agents employed—no matter whether phagocytes, opsonins, bacteriolysins, lactic acid toxines, or chemical agents—shall be brought into intimate contact with the micro-organism to be destroyed. Now from the examination of a fatal case of enteric fever recently made by Dr. Josef Koch, it appears that in certain cases the gall-bladder is thickened and filled with papillomatous processes containing homogeneous masses surrounded by necrotic areas. The necrotic areas sometimes extended through the whole thickness of the papillæ, finally bursting and emptying their contents into the interior of the gall-bladder. The homogeneous masses thus extruded into the gall-bladder were found to be collections of typhoid bacilli, the toxines of which had killed the surrounding tissues. We do not yet know whether similar changes occur in the typhoid carrier, but it is extremely probable that they do, and that the bacilli in the gall-bladder, urinary passages, and intestinal wall are surrounded by areas of dead tissue through which no curative agent can penetrate. It follows, therefore, that no cure can result in such cases until all the contents of the necrotic areas have been discharged, which may be the work of years.

It is also plain that if the "typhoid carrier" is to have a speedy cure he must be detected before these chronic changes have been produced in his tissues. Consequent on the installation of the depôts at Naini Tal, and elsewhere in India, the convalescents from enteric fever are examined soon after the cessation of the fever, and from this policy will result an early diagnosis of the carrier condition, which cannot fail to be of benefit not only to the individual, but to the State.

SUMMARY OF SOME RECENT OUTBREAKS OF ENTERIC FEVER SUPPOSED TO BE CAUSED BY "CARRIERS," AND DEDUCTIONS THEREFROM.

It may be of interest to describe briefly some notable outbreaks of enteric fever which have been investigated recently, and which appear to have originated from definite typhoid carriers.

Series of Cases of Enteric Fever Apparently Caused by a Carrier.*

(*Reported by Dr. Soper.*)

In June, 1902, the carrier, who was a cook, joined a family three weeks before the summer exodus. At the end of this time enteric fever appeared. In October, 1903, she entered the service of another family. Nine months later—viz., June, 1904—the family went for the summer holiday, taking the cook with them. Arrived at their summer residence, the household visited had an outbreak of enteric fever. The cook found a new situation, and in September, 1906, she caused another outbreak of enteric fever shortly after she arrived; while in January, 1907, another family owed its attack of enteric fever to her entry into the house.

Outbreak of Enteric Fever at the Brentry Reformatory, Bristol.*

The home contains 240 inmates and 24 resident officers. It has been open since 1899, and enteric fever first appeared in 1906. There were no cases of enteric fever in the neighbourhood. In September, 1906, a kitchen helper developed enteric fever, and in November three more cases occurred. In January, 1907, a fourth case occurred. Then there was a lull until May, 1907, when fresh cases occurred, continuing through July, August, September, October and November, in small groups and at varying intervals. By excluding other sources of infection the cause of the outbreak was supposed to be a "carrier" case dealing with the milk of the institution. It was then ascertained that an inmate employed as cook and dairymaid, Mrs. H., had suffered in

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* Extract from *Lancet*, November 28th, 1908.

January, 1901, from a severe attack of enteric fever. On November 13th she was isolated as a suspect, until arrangements could be made for the examination of her excreta. The last case of enteric fever occurred on November 25th, twelve days after her isolation commenced; and the institution has remained free up to the present time. On December 20th the *B. typhosus* was isolated from the excreta of Mrs. H. The belief that Mrs. H. was the actual cause of the Brentry outbreak, if not proved by the foregoing sequence of events, at least provided a working hypothesis which stood the test of experiment.

Outbreak of Enteric Fever at Grove House Home, Brislington, near Bristol.*

In 1904 this home, opened in 1899, was occupied by thirty-six girls boarded out by the Bristol Guardians. Enteric fever first appeared in the third week of May, and from that time until the end of September, when the home was closed, cases continued to occur in crops at intervals of a week or more. Twenty-five cases developed enteric fever, eight other suspected cases occurred and two deaths resulted. How the first case arose it was found impossible at the time to ascertain; there had been no enteric fever in the district for months. Every possible means was taken to control the disease by disinfection, boiling of milk, cleanliness, and precautions as to food, all without avail. The medical officer suspected a human "carrier," and the girls, *but not the staff*, were systematically examined. Still the outbreak went on and did not cease until the home was closed at the end of September. The cook then left to take a private situation. This history might have been held to afford a fairly complete example of an outbreak of enteric fever due to "insanitary" local conditions. It now appears, however, that on February 3rd, 1904, Mrs. H., *the same woman who was in* 1906 *cook and dairymaid at Brentry*, had been transferred from the Bristol workhouse to assist at Grove House in the kitchen. On May 2nd she was taken on as a permanent servant, and her duties were to assist in the kitchen and attend to the boiling of the milk and its preparation for consumption by the children. She left to take a private situation on September 2nd, and the last case of enteric fever was notified twenty days later, the home being closed at the end of September.

An Outbreak in a Detachment of the Bedfordshire Regiment at Kasauli.

During the month of August, 1907, five cases of enteric fever occurred in this detachment. Four of the men were admitted into the Station Hospital, Kasauli, and cultivations from the blood proved that they were infected by the *B. typhosus*. One man left Kasauli and was admitted into hospital at Jhansi on August 2nd suffering from enteric fever. So that five men of the detachment acquired the infection about the same time in the same place. The disease did not extend to any other units at Kasauli. Each case occurred in a different room of the two barracks in which the detachment was quartered, so that there was no direct personal communication between the patients. The two barracks were supplied with food from a common cook-house and they had a common latrine and urinal.

In order to determine the origin of the outbreak an examination of the blood, urine, and fæces of all the cooks and "contacts" was made, and a cook of the detachment was found excreting the *B. typhosus* in large numbers in his fæces. The cook was at once isolated and no further case of enteric fever occurred in this unit.

Two Cases of Enteric Fever caused by a "Chronic Carrier."

In July, 1906, a boy (Joseph T.) was admitted to the Station Hospital, Kasauli, from the married quarters, suffering from enteric fever. Later in the month a next-door neighbour of this family, Mrs. C., was admitted with enteric fever. In order to trace the source of these two cases the blood, urine, and fæces of all the members of the T. family were examined. The result of this investigation showed that Mrs. T., the mother of the first patient, was excreting *B. typhosus* in her fæces.

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* Extract from *Lancet*, November 28th, 1908.

A Chronic Bacillus Carrier in the Scottish Rifles, and in this Unit a considerable number of Cases of Enteric Fever occurred.

On examining a large number of healthy soldiers at Meerut, from the different units stationed there, one man was found excreting the *B. typhosus* in his fæces. He belonged to the Scottish Rifles, and it is a noteworthy fact that seven out of ten cases of acute enteric fever in Meerut came from this unit.

A Series of Cases of Enteric Fever Apparently Caused by a "Carrier."

During the first three weeks of September, 1908, four cases of enteric fever were admitted to hospital at Aldershot, all of which came from C and D Companies of the same regiment—1st Battalion East Kent Regiment—which was quartered in Badajos Barracks. On October 10th the 3rd Battalion Rifle Brigade, stationed at Bordon Camp, sent 123 N.C.Os. and men into Aldershot. This detachment was quartered in Badajos Barracks, and of the five rooms handed over to the men, two rooms had been previously occupied by the men of C and D Companies of the East Kent Regiment. On November 3rd and following days, four cases of enteric fever occurred amongst the men of the Rifle Brigade who had occupied the rooms used by C and D Companies of the East Kent Regiment, and two other cases of enteric fever from this regiment were admitted to hospital at almost the same time. The men of C and D Companies were then examined, and Corporal (now Lance-Serjeant) I. was found to be excreting the *B. typhosus* in his urine.

On enquiring into the previous history of this man it was ascertained that he had suffered from enteric fever in Aden in 1904; the same year he came to Dover, and three men in his company developed enteric fever. In 1907 he came to Longmoor Camp, and two men in his company there acquired enteric fever.

After the isolation of Lance-Serjeant I. no further cases of enteric fever occurred in the Badajos Barracks.

On considering the evidence as to the "typhoid carrier" being the cause of these outbreaks, attention should be directed to the following points:—

(A) A carrier case has been present in each outbreak, and, on isolating this individual, no further cases of enteric fever have developed.

(B) When the carrier case has been employed in the preparation of food, and especially in handling milk, it is easy to understand that a definite, though probably small, quantity of infective material may gain access to the food supplies. At the same time it must be admitted that examination of the hands of carriers, except immediately after the act of micturition, have often failed to demonstrate the presence of the infecting microbe. Also there are cases on record where a typhoid carrier has been employed as a cook without any outbreak of enteric fever occurring amongst the men supplied with food by him.

(C) When the carrier is not employed in connection with the preparation of food, it is conceivable that the infection might be carried: (1) by direct personal contact; (2) through the agency of latrines and urinals, either by dust or by flies.

As regards direct personal contact there is little evidence that, in peace time, this mode of infection is common in the army. Cases supposed to be due to carriers have rarely occurred in adjacent beds, or even in the same barrack room. It is, however, probable that in camp life, and especially on active service, when men are crowded in tents in actual bodily contact, such a mode of infection might hold a prominent place.

As regards infection from urinals and latrines there has been a definite diminution in the number of cases of enteric fever in certain stations in India following on efficient sanitary care of these appliances and the substitution of disinfecting solutions, with a consequent disappearance of flies, for dry earth. It does not, of course, follow that the excreta of the carrier cases have been the only infecting agents in these stations; it is more than probable that the excreta of undiagnosed and ambulatory cases of enteric fever have also played a part in such infection.

(D) Outbreaks of enteric fever have a marked seasonal incidence, and to explain this we must believe either that the carrier has a corresponding seasonal excretion of typhoid bacilli, or that under climatic and other conditions there is a seasonal wave in the virulence of the bacilli.

The work carried out in the Royal Army Medical College has shown that in some carriers there is neither a special seasonal excretion nor a seasonal virulence of typhoid bacilli. Much more work must be done before definite statements can be made on these points.

(E) Since the establishment of the Naini Tal Depôt for enteric convalescents, it has been found that the admissions for enteric fever from all stations which send convalescents there show a reduction of 9 per cent. on the figures for 1907, while the remaining stations show an increase of 26·6 per cent.

Taking all these points into consideration, the Advisory Board believe that there is sufficient evidence to justify the policy pursued, and recommend that "since arrangements are now being made for early treatment on discovery of these cases, it is desirable that any man ascertained to be a carrier should, after a period of observation in England not exceeding three months, be discharged from the Service, unless he elects to remain in hospital for treatment. When any man is discharged the Medical Officer of Health for the district in which he will reside should be notified."

This recommendation has been approved by the Army Council.

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